Rare disease

A rare case of portal vein gas: accidental hydrogen peroxide ingestion

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Summary

Hydrogen peroxide (H_2O_2) is a colourless and odourless liquid with oxidant characteristics used for various purposes. Whereas in lower concentrations (3%), H_2O_2 is used as a disinfectant in home cleaning products and wound care, in higher concentrations (35%) it is used in textile and paper industry as a bleaching agent and is diluted for use in lightening hair dyes. Like other caustic substances, direct injuries may develop if H_2O_2 is swallowed and systemic air embolisms may occur due to the resultant gaseous oxygen. This study discusses a patient who was detected with the presence of gas in the portal venous system due to H_2O_2 intoxication and was treated conservatively.

BACKGROUND

Hydrogen peroxide (H_2O_2) ingestion causes severe toxicities including death. Not being fatal in adults, cerebral infarction due to gaseous oxygen occurring after ingestion and neurological deficits may develop.¹ After oxygen enters the circulation, gas may occur in the portal vein whether the mucosa is damaged or not.² Moreover, H_2O_2 can directly enter the portal circulation after being absorbed by the stomach wall and can break down into water and oxygen in circulation.³ Venous embolisations occur when the amount of oxygen in the blood exceeds maximal solubility. It can also cause mesenteric ischaemia with a mortality rate reaching occasionally up to 75–90%.⁴ In H_2O_2 intoxication, knowing the underlying cause and patient's clinical status guides the treatment and this is more important than the presence of gas in the portal vein.⁴

The diagnosis of hepatic portal venous gas (HPVG) is usually made by plain abdominal radiography, sonography, colour Doppler flow imaging or CT scan. HPVG is not by itself a surgical indication and the treatment depends mainly on the underlying disease. The prognosis is related to the pathology itself and is not influenced by the presence of HPVG.⁵

CASE PRESENTATION

A 20-year-old male patient experienced burning in his throat after accidentally drinking two sips (approximately 30 cc) from a colourless and odourless liquid. It was determined that the liquid the patient drank was a H_2O_2 solution in 30% concentration used by his wife to bleach her hair. Thirty minutes after the liquid intake, following nausea and vomiting, a small amount of blood came out of the patient's mouth and they consulted an emergency service. The patient was given 500 ml milk to drink and then was sent to our hospital for further examination and treatment. He was conscious and cooperative when he first came to our

hospital. Burning in the mouth and throat, nausea and chest and epigastric region pain were the symptoms. The blood pressure, pulse, respiration rate, fever and oxygen saturation were found to be 130/70 mm Hg, 52/min, 16/min, 36.2°C and 98%, respectively. There were hyperaemia in tongue, mouth mucosa and oropharynx and sensitisation on the epigastric region during physical examination. Cardiopulmonary and neurological examinations were normal. The performed posterior–anterior chest radiography and electrocardiography were assessed to be normal. White blood cell was 17 700/mc/l in terms of the laboratory; the other parameters were within normal limits. The patient's oral intake was ended in emergency; intravenous fluid treatment was initiated with 40 mg intravenous omeprazole in 10 mg/h infusion doses and 10 mg intravenous metoclopramide 2 × 1. Computed tomographies of the thorax and abdomen were performed after discomfort started in the patient's chest and pain on the epigastric region did not decrease. Paraesophageal and paraaortic air densities on superior mediastinum in computed thorax tomography (figure 1) and widespread air densities in portal venous branches of the liver in computed abdomen tomographies (figure 2) were observed. Oesophagoscopy was performed on the patient by administering an oral contrast substance (*Meglumine diatrizoate*, 50 ml) on suspicion of oesophagus perforation; it was observed that the contrast substance did not leak out of the lumen (figure 3A,B). Immediate surgical intervention was not considered for the patient and treatment with teicoplanin 400 mg 2 × 1, sulbactam/cefoperazone 1 g 3 × 1, ornidazole 500 mg 2 × 1, pantoprazol 40 mg 1 × 1 by intravenous administration was initiated and an upper gastrointestinal system endoscopy was planned for 72 h later. In control tomographies performed 6 h later, no increase was observed in mediastinal air, but an essential decrease was observed in air densities of liver portal venous branches (figure 4). Partial peeling on mucosa

Figure 1 Paraesophageal (black arrow) and paraaortic (white arrow) air densities in mediastinum.

starting from the upper oesophageal sphincter and continuing down to the lower end and ulcerous lesions covered with white exudates the largest of them in 4 mm diameter (2a burn of upper gastrointestinal), hyperaemia and oedema in fundus, corpus and antrum mucosa of the stomach were detected in the patient's upper gastrointestinal system endoscopy. At the end of the third day, oral fluid intake was started. No new symptoms or complications developed during the patient's emergency follow-ups; white cell count dropped to 9800 mc/l. The patient was discharged at the end of the fourth day. He did not come for the controlling of the upper gastrointestinal system endoscopy.

DIFFERENTIAL DIAGNOSIS

Trauma-related mucosal injury, intestinal obstruction, endoluminal procedures, infection or inflammatory bowel disease.

TREATMENT

- ▶ Conservative treatment
- ▶ Surgery treatment
- ▶ Hyperbaric oxygen therapy.

OUTCOME AND FOLLOW-UP

The presence of air in the portal venous system due to drinking H_2O_2 can be conservatively treated unlike other clinical conditions that cause air presence in portal veins, as long as there is no perforation in the gastrointestinal system.

Figure 2 Unenhanced CT scan of the abdomen shows widespread air densities in portal venous branches (black arrows).

Figure 3 (A, B) Oesophagoscopies with oral contrast shows no contrast substance leakage out of the lumen.

DISCUSSION

H_2O_2 forms in all cells as a product of oxidative metabolism and is intensively decomposed to water and molecular oxygen with catalase enzyme found in the liver and erythrocytes. The toxic effects of H_2O_2 basically consist of local tissue damage and gas formation.⁶ Whereas often nausea, vomiting, haematemesis and distension in the abdomen develop depending on gastrointestinal system irritation as a result of drinking H_2O_2 in lower concentrations, systemic findings and death can be seen in higher concentration intoxications.^{7 8} One millilitre of H_2O_2 in 30% concentration exothermically reacts with the catalase enzyme found in tissues under normal temperature and pressure values and produces approximately 100 ml oxygen gas.³ Extreme amounts of oxygen gas accumulated in closed body cavities can lead to mechanical stress on hollow organs and, as a consequence, lead to perforations.⁹ Although the presence of gas in the portal venous system can be encountered in many clinical conditions, it is a finding that indicates the existence of severe intra-abdominal pathologies that require immediate surgical intervention often, such as intestinal ischaemia and mesenteric vascular incidents.¹⁰ Conservatively treated cases where air was detected in portal veins in addition to gastrointestinal system irritation findings following the intake of H_2O_2 in higher concentrations have been reported.^{11 12} Moon *et al*¹³ reported a case where haemorrhagic gastritis developed and air was detected in portal veins following oral intake of H_2O_2 in a lower concentration (3%). Ghai *et al*¹¹ identified widespread gas in the portal vein and gastric wall thickening in a 31-year-old female patient who drank 35% H_2O_2 , who completely recovered on the third day. Luu *et al*³ and de

Figure 4 Unenhanced CT scan of the abdomen obtained at sixth hour of admission shows the resolution of air densities in portal venous branches.

Merlier and Gilbeau¹⁴ have each reported similar cases as well. In the patient we reported, we discovered air in the portal venous system following intake of H₂O₂ in 30% concentration and this gas completely recovered on the third day. Our patient was discharged with full recovery on the fourth day. Ghai and O'Malley¹¹ have stated, in the differential diagnosis of gas presence in the portal vein due to H₂O₂ intake, that trauma-related mucosal injury, intestinal obstruction, endoluminal procedures, infection or inflammatory bowel disease must be considered. The gold standard for safely assessing the depth, extent of injury and appropriate therapeutic regimen is oesophagogastroduodenoscopy. Over 80% of patients with grade 3 burns develop stricture formation, while one-third of those with grade 2 develop pyloric stenosis, acid regurgitation and perforation.¹⁵ Cheng *et al* have reported less gastrointestinal complications. In that study, only 50% of patients with grade 3 burns developed stricture formation, while 10% of those with grade 2 developed gastrointestinal complication. These lower results are thought to be because of the development and use of more effective antiacid medications (proton pump inhibitors, H₂ antagonists) and more aggressive use of nasogastric irrigation to reduce the effect of the substance ingested.¹⁶

In the literature, patients who developed somnolence, epileptic seizures and left hemiparesis as a result of drinking H₂O₂ and detected with air emboli were successfully treated with hyperbaric oxygen therapy.^{17–20} French *et al*¹⁹ treated 11 patients with portal venous gas embolisms due to H₂O₂ with hyperbaric oxygen therapy successfully. Also, cases where arterial and venous embolisms and cerebral air embolism developed as a result of using H₂O₂ locally in wound debridement and surgical site cleaning have been reported.^{21–22} As there was no neurological deficit in our patient, cerebral air embolism was not considered. The centre that we work in does not have a hyperbaric oxygen therapy system, so it was not performed to our patient.

In conclusion, drinking H₂O₂ may cause the generation of air in the portal venous system besides creating gastrointestinal system irritation-related symptoms.

Learning Points

- The presence of air in the portal venous system due to drinking H₂O₂ can be conservatively treated unlike other clinical conditions that cause air presence in portal veins, as long as there is no perforation in the gastrointestinal system.
- Cerebral air embolism should come to mind if neurological findings are observed following intake of H₂O₂ and hyperbaric oxygen therapy should be considered as the treatment.

Competing interests None.

Patient consent Obtained.

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